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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
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NEWS 12	Apr 08	"Ask CAS" for self-help around the clock
NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available

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=> s il-6 (p) antibod? (p) uveitis

L1 19 IL-6 (P) ANTIBOD? (P) UVEITIS

=> dup rem l1

PROCESSING COMPLETED FOR L1
L2 7 DUP REM L1 (12 DUPLICATES REMOVED)

=> d l2 total ibib kwic

L2 ANSWER 1 OF 7 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002131940 MEDLINE
DOCUMENT NUMBER: 21856611 PubMed ID: 11867593
TITLE: Inhibitory effects of pyrrolidine dithiocarbamate on endotoxin-induced uveitis in Lewis rats.
AUTHOR: Ohta Kouichi; Nakayama Kohzo; Kurokawa Toru; Kikuchi Takanobu; Yoshimura Nagahisa
CORPORATE SOURCE: Department of Ophthalmology, Shinshu University School of Medicine, 3-1-1 Asahi, Matsumoto 390-8621, Japan.. kohta@hsp.md.shinshu-u.ac.jp
SOURCE: INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE, (2002 Mar) 43 (3) 744-50.
Journal code: 7703701. ISSN: 0146-0404.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020228
Last Updated on STN: 20020403
Entered Medline: 20020328

AB . . . of pyrrolidine dithiocarbamate (PDTC), an antioxidant nuclear factor (NF)-kappaB inhibitor, on the ocular inflammation induced by lipopolysaccharide (LPS). METHODS: Endotoxin-induced **uveitis** (EIU) was produced by a footpad injection of 200 microg LPS in male Lewis rats. PDTC (200 mg/kg) was injected. . . . concentration in the aqueous humor (AqH) was determined from the AqH collected at 24 hours. Immunohistochemical staining with a monoclonal **antibody** against activated NF-kappaB was performed to evaluate the effect of PDTC on NF-kappaB activation. Interleukin (IL)-1beta, **IL-6**, and tumor necrosis factor (TNF)-alpha mRNA expression in the iris-ciliary

body (ICB) was determined by RNase protection assay (RPA). The. . . ICB was reduced by the PDTC treatment. The ICB at 6 hours after LPS injection exhibited increased expression of IL-1beta, **IL-6**, and TNF-alpha mRNAs, which was decreased after PDTC pretreatment. PDTC also significantly diminished the levels of these cytokines and nitrite-nitrate. . .

L2 ANSWER 2 OF 7 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000396206 MEDLINE
DOCUMENT NUMBER: 20391350 PubMed ID: 10937571
TITLE: IL-6 antagonizes TGF-beta and abolishes immune privilege in eyes with endotoxin-induced uveitis.
AUTHOR: Ohta K; Yamagami S; Taylor A W; Streilein J W
CORPORATE SOURCE: Schepens Eye Research Institute and Department of Ophthalmology, Harvard Medical School, Boston, Massachusetts 02114-0115, USA.
CONTRACT NUMBER: EY05678 (NEI)
SOURCE: INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE, (2000 Aug) 41 (9) 2591-9.
Journal code: GWI; 7703701. ISSN: 0146-0404.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000824
Last Updated on STN: 20000824
Entered Medline: 20000815

AB PURPOSE: To determine the immunosuppressive status of aqueous humor (AqH) from mouse eyes afflicted with endotoxin-induced **uveitis** (EIU) and to identify the relevant cytokines responsible for immunomodulatory activity within EIU AqH. METHODS: Bacterial lipopolysaccharide (LPS) was injected. . . 12, 24, and 48 hours, was evaluated for content of transforming growth factor (TGF)-beta, tumor necrosis factor (TNF)-alpha, interleukin (IL)-1beta, **IL-6**, and interferon (IFN)-gamma and capacity to suppress anti-CD3-driven T-cell proliferation.
Cytokine mRNA expression in iris-ciliary body (I/CB) was analyzed by. . . increased even though the fluid lost its capacity to suppress T-cell activation. At this time, AqH contained high levels of **IL-6**, and I/CB contained high levels of **IL-6** mRNA. When **IL-6** was neutralized with specific **antibodies**, inflamed AqH reacquired its capacity to suppress T-cell activation, which correlated with high levels of TGF-beta. Coinjection of **IL-6** plus antigen into the anterior chamber of the eye of normal mice prevented antigen-specific anterior chamber-associated immune deviation (ACAID). CONCLUSIONS: LPS-induced intraocular inflammation is associated with local production of **IL-6**, which robs AqH of its immunosuppressive activity, perhaps by antagonizing TGF-beta. The fact that **IL-6** antagonized ACAID induction in normal eyes suggests that strategies to suppress the intraocular synthesis of **IL-6** may reduce inflammation and restore ocular immune privilege.

L2 ANSWER 3 OF 7 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1998149477 MEDLINE
DOCUMENT NUMBER: 98149477 PubMed ID: 9489833
TITLE: Prolactin and interleukin 6 in prepubertal girls with juvenile chronic arthritis.
AUTHOR: Picco P; Gattorno M; Buoncompagni A; Facchetti P; Rossi G; Pistoia V
CORPORATE SOURCE: 2nd Division of Pediatrics, Giannina Gaslini Scientific Institute, Genoa, Italy.
SOURCE: JOURNAL OF RHEUMATOLOGY, (1998 Feb) 25 (2) 347-51.

Journal code: JWX; 7501984. ISSN: 0315-162X.
PUB. COUNTRY: Canada
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980625
Last Updated on STN: 19980625
Entered Medline: 19980616

AB . . . (JCA) and with previous acute postinfectious arthritis in remission (AA), and to correlate the relationship of PRL versus interleukin 6 (**IL-6**) serum levels. METHODS: Eleven girls with antinuclear **antibody** (ANA) positive early onset pauciarticular JCA, 8 with ANA negative late onset pauciarticular JCA of various forms (considered to have spondyloarthropathy, SpA), and 7 who had had AA were evaluated for serum concentrations of PRL, **IL-6**, and thyroid hormones and presence of **uveitis**. All were prepubertal and without clinical or biological signs of disease activity. RESULTS: Mean serum concentrations of PRL were significantly. . and p = 0.025, respectively) and to controls. Both ANA positive and SpA patients showed increased mean serum concentrations of **IL-6** in comparison with AA patients and controls. Significant correlation between PRL and **IL-6** concentrations (r = 0.604, p = 0.002) was observed from the whole series. CONCLUSION: We found a direct correlation between serum levels of PRL and **IL-6** in both ANA positive JCA patients and in ANA negative SpA patients; thus, hyperprolactinemia correlates better with the chronic course. . .

L2 ANSWER 4 OF 7 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1998001501 MEDLINE
DOCUMENT NUMBER: 98001501 PubMed ID: 9343336
TITLE: Elevated interleukin 6 activity in aqueous humor of cats with uveitis.
AUTHOR: Lappin M R; Dow S W; Reif J S; Chavkin M J
CORPORATE SOURCE: Department of Clinical Sciences, College of Veterinary Medicine, Colorado State University, Fort Collins 80523, USA.
SOURCE: VETERINARY IMMUNOLOGY AND IMMUNOPATHOLOGY, (1997 Aug) 58 (1) 17-26.
Journal code: XCB; 8002006. ISSN: 0165-2427.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971208

AB The purpose of this study was to assess the role of interleukin 6 (**IL-6**) in feline **uveitis** by measuring **IL-6** activity in the serum and aqueous humor of cats. Serum and aqueous humor was collected from clinically normal, random source. . . experimentally inoculated with Toxoplasma gondii strain ME49 and sampled sequentially for 20 months (n = 4); and client-owned cats with **uveitis** (n = 27). Interleukin 6 activity was measured in each sample. Client-owned cats with **uveitis** were also evaluated for evidence of present or prior exposure to T. gondii, feline leukemia virus, feline immunodeficiency virus, and. . . aqueous humor of clinically normal cats. Interleukin 6 activity was detected in 22/27 (81.5%) aqueous humor samples from cats with **uveitis**, with a

range of 28.9 U ml(-1)-15702.9 U ml(-1) (mean = 1911.9 U ml[-1], SD = 3946.7 U ml[-1]). Serologic. . . exposure to T gondii, feline immunodeficiency virus, feline leukemia virus, or a coronavirus was present in 21/27 (77.8%) cats with **uveitis**. Interleukin 6 was detected in the aqueous humor of 18/21 (85.7%) and 3/6 (50%) of the cats with and without serologic evidence of exposure to one to the infectious diseases, respectively. Statistically significant increases in mean **IL-6** activity in aqueous humor were found for cats with any evidence of infection with T. gondii, for cats with T. gondii antigen in aqueous humor and for cats with coronavirus **antibody** titers > or = 1:100. Aqueous humor **IL-6** activity was greater than corresponding serum **IL-6** activity in 21/27 cats.These results show that **IL-6** is produced intraocularly in some cats with **uveitis** and that **IL-6** may be a mediator of **uveitis** in cats.

L2 ANSWER 5 OF 7 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 96238054 MEDLINE
DOCUMENT NUMBER: 96238054 PubMed ID: 8647191
TITLE: IL-12 inhibits endotoxin-induced inflammation in the eye.
AUTHOR: Whitcup S M; Rizzo L V; Lai J C; Hayashi S; Gazzinelli R; Chan C C
CORPORATE SOURCE: National Eye Institute, National Institutes of Health, Bethesda, MD 20892-1858, USA.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1996 May) 26 (5) 995-9. Journal code: EN5; 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19960805
Last Updated on STN: 19960805
Entered Medline: 19960725

AB . . . was to examine the importance of IL-12 in endotoxin-induced ocular inflammation. The number of inflammatory cells infiltrating eyes with endotoxin-induced **uveitis** (EIU) was significantly increased in animals treated with intraperitoneal anti-IL-12 **antibody** when compared to control animals, but there was no difference in infiltrating inflammatory cells in the eyes of animals treated. . . Cytokine analysis of the aqueous humor obtained from eyes with EIU showed increased levels of IFN-gamma and decreased levels of **IL-6** in eyes receiving intraocular IL-12. These data show that IL-12 has an inhibitory effect on endotoxin-induced inflammation in the eye. . .

L2 ANSWER 6 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 96271362 EMBASE
DOCUMENT NUMBER: 1996271362
TITLE: [Endotoxin-induced uveitis prevention with human immunoglobulin preparations for intravenous use (IVIg)]. PREVENCION DE LA UVEITIS INDUCIDA POR ENDOTOXINA CON PREPARACIONES DE INMUNOGLOBULINAS HUMANAS PARA USO INTRAVENOSO (IVIG).
AUTHOR: Obrador E.; Peinado E.J.; Kozak Y.; Ruiz-Moreno J.M.; Alio J.L.
CORPORATE SOURCE: Division de Oftalmologia, Facultad de Medicina, Universidad de Alicante,03690 Alicante, Spain
SOURCE: Archivos de la Sociedad Espanola de Oftalmologia, (1996) 71/2 (133-140). ISSN: 0365-6691 CODEN: ASEOAK
COUNTRY: Spain
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
012 Ophthalmology

029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish
AB Several cytokines are released during Endotoxin-Induced-**Uveitis** (EIU), especially interleukin-1 (IL-1), interleukin-6 (**IL-6**), and tumour necrosis factor-alpha (TNF-.alpha.). In vitro experiments have shown a suppression of IL-1 and TNF-.alpha. production by rabbit peritoneal. . . blockade of Fc receptors on cells of the reticulo-endothelial system thus interfering with cytokine production, or the V region (anti-cytokines **antibodies** present in IVIg could neutralize the cytokines induced by LPS injection).

L2 ANSWER 7 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 95084974 EMBASE
DOCUMENT NUMBER: 1995084974
TITLE: Immunohistochemical and cytokine analysis of eyes from rats with adjuvant arthritis.
AUTHOR: Murray P.I.; Rene C.; Southwood T.R.; Hickton R.
CORPORATE SOURCE: Academic Unit of Ophthalmology, Birmingham and Midland Eye Hospital, Church Street,Birmingham B3 2NS, United Kingdom
SOURCE: Ocular Immunology and Inflammation, (1995) 3/1 (15-22). ISSN: 0927-3948 CODEN: OIINEN
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 012 Ophthalmology
031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Adjuvant induced arthritis in the rat is an animal model of juvenile chronic arthritis (JCA). An anterior **uveitis** may also develop in this model which closely resembles that seen in association with JCA in the human. Female Lewis. . . were examined by slit-lamp biomicroscopy every two days, and the animals sacrificed 21 days post-immunisation. Aqueous humour and serum interleukin-6 (**IL-6**) levels were determined using the B9 bioassay, and serial tissue sections of the globes were stained with a variety of monoclonal **antibodies**. In the FCA group, arthritis was detected in two rats, but no rats from either group developed clinical **uveitis**. Immunohistochemical analysis revealed greater numbers of macrophages and MHC class II+ve cells in the irises and ciliary bodies of FCA treated rats than were seen in the FIA group. No other cellular infiltrate was detected. Serum **IL-6** levels were greater in the FCA group (range 6-122 U/ml, median 42) than the FIA group (range 2-27, median 3). . . the highest values seen in the two rats which developed arthritis (100, 122 U/ml). In the aqueous of both groups, **IL-6** levels were < 5 U/ml, and albumin levels were not significantly different (FCA median = 2.0 mg/ml, FIA median =. . . in the adjuvant induced arthritis model, infiltration with macrophages and MHC class II+ve cells occurs in the absence of clinical **uveitis**.

=> s anti-il-6 (p) antibod?

L3 1738 ANTI-IL-6 (P) ANTIBOD?

=> s anti-il-6 (p) antibod? (p) sclerosis

L4 23 ANTI-IL-6 (P) ANTIBOD? (P) SCLEROSIS

=> dup rem l4

PROCESSING COMPLETED FOR L4
L5 8 DUP REM L4 (15 DUPLICATES REMOVED)

=> d l5 total ibib kwic

L5 ANSWER 1 OF 8 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999242622 MEDLINE
DOCUMENT NUMBER: 99242622 PubMed ID: 10225968
TITLE: Endogenous IL-1alpha from systemic sclerosis fibroblasts induces IL-6 and PDGF-A.
AUTHOR: Kawaguchi Y; Hara M; Wright T M
CORPORATE SOURCE: Division of Rheumatology and Clinical Immunology, Department of Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania 15213,. USA.YHK02262@niftyserve.ne.jp
CONTRACT NUMBER: AR-44266 (NIAMS)
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (1999 May) 103 (9) 1253-60.
Journal code: HS7; 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990618
Last Updated on STN: 19990618
Entered Medline: 19990610
AB It is reported that fibroblasts derived from clinically affected skin areas of patients with systemic **sclerosis** (SSc) have the ability to overproduce several cytokines and growth factors (i.e., IL-6, PDGF), an ability that might be involved. . . led to decreased levels of IL-6 and PDGF-A expression in SSc fibroblasts. Moreover, the blocking of the IL-6 response using **anti-IL-6 antibody** resulted in a significant reduction of procollagen type I in cultured SSc fibroblasts. These results suggest that endogenous IL-1alpha expressed. . .

L5 ANSWER 2 OF 8 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998307525 MEDLINE
DOCUMENT NUMBER: 98307525 PubMed ID: 9645618
TITLE: IL-6-deficient mice are resistant to the induction of experimental autoimmune encephalomyelitis provoked by myelin oligodendrocyte glycoprotein.
AUTHOR: Okuda Y; Sakoda S; Bernard C C; Fujimura H; Saeki Y; Kishimoto T; Yanagihara T
CORPORATE SOURCE: Department of Neurology, Osaka University Medical School, Suita, Japan.
SOURCE: INTERNATIONAL IMMUNOLOGY, (1998 May) 10 (5) 703-8.
Journal code: AY5; 8916182. ISSN: 0953-8178.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 19980917
Last Updated on STN: 20000303
Entered Medline: 19980908
AB . . . to wild-type mice (one out of 18 versus 17 out of 20). The delayed-type hypersensitivity response, lymphocyte proliferation response and **antibody** reactivity to MOG in IL-6-deficient mice were significantly lower than those in wild-type mice. Furthermore, the

histological examination revealed that. . . play a crucial role in the induction phase of EAE. Given the potential relevance of this animal model for multiple **sclerosis** (MS), it is possible that **anti-IL-6** therapy may be useful in the prevention of relapses of MS.

L5 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:734468 CAPLUS
DOCUMENT NUMBER: 130:181370
TITLE: Effects of interleukin-1 receptor antagonist and anti-interleukin-6 antibody on lupus nephritis in NZB/WF1 mice
AUTHOR(S): Li, Ming; Sun, Hanying; Zhang, Yunfeng
CORPORATE SOURCE: Department of Pathology, School of Basic Medical Sciences, Tongji Medical University, Wuhan, 430030, Peop. Rep. China
SOURCE: Tongji Yike Daxue Xuebao (1998), 27(4), 259-261
CODEN: TYDXEP; ISSN: 0258-2090
PUBLISHER: Tongji Yike Daxue
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB The effects of interleukin-1 receptors antagonist (IL-1ra) and anti-interleukin-6 **antibody** (**anti-IL-6**) on renal morphol. manifestation of lupus-like NZB/WF1 mice were obsd. The results showed that the lesion in IL-1ra group was focal mesenteric hyperplastic glomerular nephritis. The pathol. change was single. Moderate disseminated mesenteric hyperplastic glomerular nephritis was obsd. in **anti-IL-6** group, while in the control group, the pathol. manifestation was severe disseminated mesenteric hyperplastic glomerular nephritis with obvious multiple- change lesion. The electron microscope showed that the multiple electro- dense deposition was obsd. in the glomerulus in all the three groups. The lesion in IL-1ra group was small and in less amt., while in the control group was big and in larger amt. The results suggest that IL-1ra can obviously alleviate the glomerular pathol. damage in lupus-like NZB/WF1 mice, which might delay the **sclerosis** of glomerulus and improve the renal function. **Anti-IL-6** also has certain improving effects on the murine glomerular lesion.

L5 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:21718 CAPLUS
DOCUMENT NUMBER: 128:126925
TITLE: Studies on synthesis of interleukin-6 and mechanism of peripheral blood B cell activation in patients with collagen disease
AUTHOR(S): Ohira, Yoko; Nishimaki, Tomoe; Kasukawa, Reiji
CORPORATE SOURCE: Dep. Intern. Med., Fukushima Med. Coll., Fukushima, 960-12, Japan
SOURCE: Fukushima Igaku Zasshi (1997), 47(4), 303-311
CODEN: FSIZAQ; ISSN: 0016-2582
PUBLISHER: Fukushima Igakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Interleukin-6 (IL-6) may have an important role for a polyclonal activation of B cells in patients with collagen disease. We examd. serum IL-6 levels and the prodn. of IL-6 by peripheral blood mononuclear cells (PBMC) from patients with systemic lupus erythematosus (SLE), mixed connective tissue disease (MCTD), rheumatoid arthritis (RA) and progressive systemic **sclerosis** (PSS). Serum IL-6 levels in patients with SLE, MCTD, RA, and PSS were significantly higher than those in normal controls. The mean levels of spontaneous secretion of IL-6 from

PBMC, monocytes and B cells were significantly higher in patients with SLE and MCTD than that in normal controls. In patients with SLE, there was a significant correlation between the level of IL-6 prodn. by PBMC and serum IgG levels, but no significant correlation with titers of anti-ss DNA **antibody**. The levels of IgG prodn. from staphylococcus aureus cowen 1 (SAC)-treated normal B cells were markedly increased with stimulation of conditioned medium of monocytes from patients with SLE. Moreover, the level of IgG prodn. from PBMC treated with **anti-IL-6** or anti-HLA-DR **antibody** was significantly decreased in patients with MCTD as compared the results without these **antibodies**. The level of U1RNP **antibody** prodn. from PBMC was also reduced by addn. of **anti-IL-6** **antibody**. These data suggest that the IL-6 stimulates the IgG prodn. via activation of B cells in patients with SLE. On the other hand, in patients with MCTD, IL-6 is thought to be produced by activated B cells following B-B cells interaction recognized with class II antigen and it stimulates prodn. of IgG and U1RNP **antibody** from B cells by autocrine mechanisms.

L5 ANSWER 5 OF 8 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96303261 MEDLINE
DOCUMENT NUMBER: 96303261 PubMed ID: 8732437
TITLE: IL-6-soluble IL-6 receptor complex inhibits the proliferation of dermal fibroblasts.
AUTHOR: Mihara M; Moriya Y; Ohsugi Y
CORPORATE SOURCE: Fuji-Gotemba Research Laboratories, Chugai Pharmaceutical Co. Ltd., Shizuoka, Japan.
SOURCE: INTERNATIONAL JOURNAL OF IMMUNOPHARMACOLOGY, (1996 Jan) 18 (1) 89-94.
Journal code: GRI; 7904799. ISSN: 0192-0561.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19980206
Entered Medline: 19970128
AB . . . investigators has reported that there is increased production of interleukin-6 (IL-6) by fibroblasts and monocytes from the patients with systemic **sclerosis** (SS). However, the precise role of IL-6 in the pathogenesis of SS remains unclear. On the basis of our previous. . . the presence of sIL-6R, dose-dependently showed much stronger suppressive effects on DF proliferation. This suppression was completely blocked by either **anti-IL-6** or anti-sIL-6R **antibody**. Furthermore, the IL-6-sIL-6R complex significantly suppressed IL-1 beta-, TNF alpha- and PDGF-AA-induced DF proliferation. These lines of evidence suggest that. . .

L5 ANSWER 6 OF 8 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96192948 MEDLINE
DOCUMENT NUMBER: 96192948 PubMed ID: 8612202
TITLE: Administration of neutralizing antibodies to interleukin-6 (IL-6) reduces experimental autoimmune encephalomyelitis and is associated with elevated levels of IL-6 bioactivity in central nervous system and circulation.
AUTHOR: Gijbels K; Brocke S; Abrams J S; Steinman L
CORPORATE SOURCE: Department of Neurology and Neurological Sciences, Stanford University School of Medicine, CA 94305-5429, USA.
CONTRACT NUMBER: NS18235 (NINDS)
SOURCE: MOLECULAR MEDICINE, (1995 Nov) 1 (7) 795-805.

Journal code: CG3; 9501023. ISSN: 1076-1551.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960613
Last Updated on STN: 20000303
Entered Medline: 19960606

AB . . . (IL-6) in the central nervous system (CNS) in experimental autoimmune encephalomyelitis (EAE), an animal model for the human disease multiple **sclerosis**. MATERIALS AND METHODS: To assess the role of IL-6 in autoimmune CNS inflammation, we administered neutralizing **antibodies** to IL-6 in the EAE model. Their effect was examined at the clinical and histopathological level. Levels of administered **antibody** and IL-6 bioactivity were followed in serum and cerebrospinal fluid (CSF). RESULTS: Systemically administered **antibodies** penetrated into the fluid CSF in animals in which EAE was induced. Administration of **anti-IL-6** reduced the development of actively induced as well as adoptively transferred EAE and was associated with increased levels of IL-6 activity in the CSF and to a lesser extent in the serum. **Anti-IL-6** was still effective when given 1 day before the onset of disease signs in adoptively transferred EAE. The disease-reducing effect of **anti-IL-6** was also reflected at the pathological level by the absence of inflammatory infiltrates in the CNS. CONCLUSIONS: Our study indicates. . . an important role in autoimmune CNS inflammation. However, due to the complex nature of the in vivo interactions of administered **antibodies**, the disease-reducing effect of the **anti-IL-6 antibodies** could be caused by neutralization of IL-6 activity or by enhancement of IL-6 activity via induction of higher IL-6 levels. . .

L5 ANSWER 7 OF 8 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 92352487 MEDLINE
DOCUMENT NUMBER: 92352487 PubMed ID: 1642659
TITLE: Anti-interleukin-6 autoantibodies in rheumatic diseases. Increased frequency in the sera of patients with systemic sclerosis.
AUTHOR: Takemura H; Suzuki H; Yoshizaki K; Ogata A; Yuhara T; Akama T; Yamane K; Kashiwagi H
CORPORATE SOURCE: Department of Rheumatology, University of Tsukuba, Japan.
SOURCE: ARTHRITIS AND RHEUMATISM, (1992 Aug) 35 (8) 940-3.
Journal code: 90M; 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199209
ENTRY DATE: Entered STN: 19920911
Last Updated on STN: 19920911
Entered Medline: 19920903

AB OBJECTIVE. To investigate the presence and the roles of anti-interleukin-6 (**anti-IL-6**) autoantibodies in rheumatic diseases, and to further elucidate clinical and pathophysiologic significance of anticytokine autoantibodies. METHODS. **Anti-IL-6** IgG autoantibodies were measured by the 125I-IL-6 binding activity of IgG, which was isolated from serum by protein A-Sepharose. RESULTS. Nine of 52 sera (17.3%) from patients with systemic **sclerosis** (SSc) contained **anti-IL-6 antibodies**, whereas only 1.9% of sera from normal subjects and 0-5% of sera from patients with other rheumatic diseases were positive for the **antibodies**. Moreover, **anti-IL-6**

autoantibodies were found predominantly among patients with the limited form of SSc (42.9%), compared with those with the diffuse form (7.9%). CONCLUSION. **Anti-IL-6** IgG autoantibodies were detected in patients with SSc, particularly those with the limited form of the disease, at a significantly increased frequency compared with normal subjects and patients with other rheumatic diseases. These results suggest that the development of **anti-IL-6** autoantibodies and IL-6 may have a role in the pathophysiology of SSc.

L5 ANSWER 8 OF 8 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 91303963 MEDLINE
DOCUMENT NUMBER: 91303963 PubMed ID: 1830124
TITLE: Distribution of interleukin-6 in normal and diseased human kidney.
AUTHOR: Fukatsu A; Matsuo S; Tamai H; Sakamoto N; Matsuda T; Hirano T
CORPORATE SOURCE: Third Department of Internal Medicine, Nagoya University School of Medicine, Japan.
SOURCE: LABORATORY INVESTIGATION, (1991 Jul) 65 (1) 61-6.
Journal code: KZ4; 0376617. ISSN: 0023-6837.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199108
ENTRY DATE: Entered STN: 19910908
Last Updated on STN: 19910908
Entered Medline: 19910816
AB . . . kidney tissue and 47 specimens obtained from diseased kidneys by biopsy were studied by indirect immunofluorescence microscopy using specific monoclonal **antibody** to human IL-6. In the normal kidney, IL-6 was distributed in the glomerular mesangial area and in vascular walls. In. . . glomerulus in various patterns. The extent of the distribution of IL-6 correlated with the average number of glomerular cells. When **sclerosis** appeared, the expression of IL-6 decreased. Occasionally, IL-6 was found in the area of synechiae or crescents as well as. . . interstitium, IL-6 was seen in atrophic tubules, and the distribution correlated with the extent of tubular atrophy. The binding of **anti-IL-6** monoclonal **antibody** to the renal sections was completely inhibited by preincubation of the **antibody** with recombinant IL-6. These results suggests that IL-6 expression is a good marker for glomerular cell proliferation and that IL-6. . .

=> s (anti-il-6 (s) receptor) (p) antibod? (p) sclerosis

L6 4 (ANTI-IL-6 (S) RECEPTOR) (P) ANTIBOD? (P) SCLEROSIS

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7 2 DUP REM L6 (2 DUPLICATES REMOVED)

=> d l7 total ibib kwic

L7 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:734468 CAPLUS
DOCUMENT NUMBER: 130:181370
TITLE: Effects of interleukin-1 receptor antagonist and anti-interleukin-6 antibody on lupus nephritis in NZB/WF1 mice

AUTHOR(S): Li, Ming; Sun, Hanying; Zhang, Yunfeng
CORPORATE SOURCE: Department of Pathology, School of Basic Medical Sciences, Tongji Medical University, Wuhan, 430030, Peop. Rep. China
SOURCE: Tongji Yike Daxue Xuebao (1998), 27(4), 259-261
CODEN: TYDXEP; ISSN: 0258-2090
PUBLISHER: Tongji Yike Daxue
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The effects of interleukin-1 **receptors** antagonist (IL-1ra) and anti-interleukin-6 **antibody** (**anti-IL-6**) on renal morphol. manifestation of lupus-like NZB/WF1 mice were obsd. The results showed that the lesion in IL-1ra group was focal mesenteric hyperplastic glomerular nephritis. The pathol. change was single. Moderate disseminated mesenteric hyperplastic glomerular nephritis was obsd. in anti-IL-6 group, while in the control group, the pathol. manifestation was severe disseminated mesenteric hyperplastic glomerular nephritis with obvious multiple- change lesion. The electron microscope showed that the multiple electro- dense deposition was obsd. in the glomerulus in all the three groups. The lesion in IL-1ra group was small and in less amt., while in the control group was big and in larger amt. The results suggest that IL-1ra can obviously alleviate the glomerular pathol. damage in lupus-like NZB/WF1 mice, which might delay the **sclerosis** of glomerulus and improve the renal function. Anti-IL-6 also has certain improving effects on the murine glomerular lesion.

L7 ANSWER 2 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 96303261 MEDLINE
DOCUMENT NUMBER: 96303261 PubMed ID: 8732437
TITLE: IL-6-soluble IL-6 receptor complex inhibits the proliferation of dermal fibroblasts.
AUTHOR: Mihara M; Moriya Y; Ohsugi Y
CORPORATE SOURCE: Fuji-Gotemba Research Laboratories, Chugai Pharmaceutical Co. Ltd., Shizuoka, Japan.
SOURCE: INTERNATIONAL JOURNAL OF IMMUNOPHARMACOLOGY, (1996 Jan) 18 (1) 89-94.
Journal code: GRI; 7904799. ISSN: 0192-0561.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19980206
Entered Medline: 19970128

AB . . . investigators has reported that there is increased production of interleukin-6 (IL-6) by fibroblasts and monocytes from the patients with systemic **sclerosis** (SS). However, the precise role of IL-6 in the pathogenesis of SS remains unclear. On the basis of our previous study showing that the complex of IL-6 and soluble IL-6 **receptor** (sIL-6R) could induce synovial fibroblast proliferation, we examined whether the IL-6-sL-6R complex could induce the proliferation of normal dermal fibroblastic. . . the presence of sIL-6R, dose-dependently showed much stronger suppressive effects on DF proliferation. This suppression was completely blocked by either **anti-IL-6** or anti-sIL-6R **antibody**. Furthermore, the IL-6-sIL-6R complex significantly suppressed IL-1 beta-, TNF alpha- and PDGF-AA-induced DF proliferation. These lines of evidence suggest that. .

.

=> s (anti-il-6 (s) receptor) (p) antibod? (p) uveitis

L8 0 (ANTI-IL-6 (S) RECEPTOR) (P) ANTIBOD? (P) UVEITIS

=> s (anti-il-6 (s) receptor) (p) antibod? (p) dermatit?

L9 0 (ANTI-IL-6 (S) RECEPTOR) (P) ANTIBOD? (P) DERMATIT?

=> s (anti-il-6 (s) receptor) (p) antibod? (p) thyroid?

L10 3 (ANTI-IL-6 (S) RECEPTOR) (P) ANTIBOD? (P) THYROID?

=> dup rem l10

PROCESSING COMPLETED FOR L10
L11 1 DUP REM L10 (2 DUPLICATES REMOVED)

=> d l11 ibib kwic

L11 ANSWER 1 OF 1 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 97281268 MEDLINE
DOCUMENT NUMBER: 97281268 PubMed ID: 9135576
TITLE: Immunoneutralization of interleukin-1, tumor necrosis factor, interleukin-6 or interferon does not prevent the LPS-induced sick euthyroid syndrome in mice.
AUTHOR: Boelen A; Platvoet-ter Schiphorst M C; Wiersinga W M
CORPORATE SOURCE: Department of Endocrinology, Academic Medical Center, University of Amsterdam, The Netherlands.
SOURCE: JOURNAL OF ENDOCRINOLOGY, (1997 Apr) 153 (1) 115-22. Journal code: I1J; 0375363. ISSN: 0022-0795.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970602
Last Updated on STN: 19970602
Entered Medline: 19970519

AB The sick euthyroid syndrome is a state of altered **thyroid** hormone metabolism which occurs during illness. The pathogenesis is incompletely understood but recent studies indicate a role of cytokines. It is unknown if cytokines released during illness are directly responsible for the changes in **thyroid** hormone metabolism. Therefore we studied if previous immunoneutralization of cytokines can prevent endotoxin (lipopolysaccharide LPS), induced sick euthyroid syndrome. LPS. . . decrease in serum triiodothyronine (T3) and thyroxine (T4). Immunoneutralization of the effects of cytokines was accomplished by administration of monoclonal **antibodies** against mouse IL-1 type-1 **receptor** (IL-1R), TNF alpha, IL-6 or interferon (IFN gamma) prior to LPS. The LPS-induced release of cytokines was affected by previous. . . anti-IL-1R did not affect serum TNF alpha but decreased serum IL-6, anti-TNF alpha decreased serum TNF alpha but not IL-6, **anti-IL-6** did not affect serum TNF alpha but hugely increased IL-6 and anti-IFN gamma decreased both serum TNF alpha and IL-6.. . . dose of immunoglobulins (1 mg), used only in the immunoneutralization of IL-6, we repeated the experiment with F(ab')2 fragments of **anti-IL-6 antibodies**. Compared with F(ab')2 fragments of control IgG, **anti-IL-6** F(ab')2 did not affect the LPS-induced rise in serum TNF alpha or the decrease in serum T3 and T4 and liver 5'-deiodinase mRNA. Serum IL-6 levels induced by LPS were, however, cleared more rapidly from the circulation when **anti-IL-6** F(ab')2 fragments rather than intact **anti-IL-6** were administered. In conclusion, immunoneutralization of IL-1, TNF alpha or

IFN gamma did not prevent the LPS-induced sick euthyroid syndrome. . .

=> s (anti-il-6 (s) receptor) (p) antibod? (p) sensitiv?

L12 40 (ANTI-IL-6 (S) RECEPTOR) (P) ANTIBOD? (P) SENSITIV?

=> dup rem l12

PROCESSING COMPLETED FOR L12
L13 15 DUP REM L12 (25 DUPLICATES REMOVED)

=> d l13 total ibib kwic

L13 ANSWER 1 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:317003 BIOSIS
DOCUMENT NUMBER: PREV200100317003
TITLE: Interleukin-6 may cause angiogenesis through VEGF induction in Castleman's disease.
AUTHOR(S): Nakahara, Hideko (1); Yoshizaki, Kazuyuki (1); Matsumoto, Tomoshige (1); Nishimoto, Norihiro (1)
CORPORATE SOURCE: (1) Department of Medical Science I, School of Health and Sport Sciences, Osaka University, Suita, Osaka Japan
SOURCE: Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 362a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000 American Society of Hematology
. ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB. . . cell type and hyaline vascular type, and mixed type is also frequently observed. We recently reported that administration of humanized **anti-IL-6 receptor** (IL-6R) **antibody**, MRA, resulted in improved CD. In addition to reduction in the size of lymph nodes, and in both the number. . . angiogenesis of CD, we have first analyzed serum levels of VEGF, IL-8, MIP-1alpha, bFGF, HGF, EGF, MCP-1, and RANTES utilizing **sensitive** ELISA system in sixteen CD patients without MRA treatment. And then, to examine if IL-6 causes angiogenesis by inducing some. . .

L13 ANSWER 2 OF 15 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999118958 MEDLINE
DOCUMENT NUMBER: 99118958 PubMed ID: 9921985
TITLE: Blocking signaling through the Gp130 receptor chain by interleukin-6 and oncostatin M inhibits PC-3 cell growth and sensitizes the tumor cells to etoposide and cisplatin-mediated cytotoxicity.
AUTHOR: Borsellino N; Bonavida B; Ciliberto G; Toniatti C; Travali S; D'Alessandro N
CORPORATE SOURCE: Istituto di Farmacologia, Policlinico P. Giaccone, Universita di Palermo, Italy.
SOURCE: CANCER, (1999 Jan 1) 85 (1) 134-44.
Journal code: CLZ; 0374236. ISSN: 0008-543X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311

Last Updated on STN: 19990311
Entered Medline: 19990224

AB . . . prostate carcinoma are poorly understood. The human prostate carcinoma PC-3 cell line, derived from a metastatic tumor and lacking androgen **receptors**, represents a useful model to investigate drug resistance. METHODS: The effects of oncostatin M (OM), antiinterleukin-6 (IL-6) treatment, or interference. . . PC-3 tumor cells to both etoposide and cisplatin. The influence of IL-6 is controlled by treating PC-3 tumor cells with **anti-IL-6** neutralizing **antibody** and, more efficiently, by a mutated IL-6, Sant7. Sant7 has a high affinity binding to the IL-6 **receptor** -alpha (IL-6Ralpha) subunit, but does not bind to the signaling subunit gp130; therefore, it behaves as a **receptor** antagonist. Both IL-6- and OM-mediated effects are inhibited by the treatment of PC-3 with an antisense oligodeoxynucleotide against gp130, the. . . the monoterpene perillic acid (PA), a posttranslational inhibitor of p21ras isoprenylation. CONCLUSIONS: These results demonstrate the protective roles in drug **sensitivity** of IL-6 and OM through signaling of the common chain gp130 and, most likely, a downstream ras-dependent pathway in PC-3. . .

L13 ANSWER 3 OF 15 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97141797 MEDLINE
DOCUMENT NUMBER: 97141797 PubMed ID: 8988055
TITLE: Interleukin-6 as a paracrine and autocrine growth factor in human prostatic carcinoma cells in vitro.
AUTHOR: Okamoto M; Lee C; Oyasu R
CORPORATE SOURCE: Department of Pathology, Northwestern University Medical School, Chicago, Illinois 60611-3008, USA.
CONTRACT NUMBER: CA 14649 (NCI)
CA33511 (NCI)
CA69851 (NCI)
SOURCE: CANCER RESEARCH, (1997 Jan 1) 57 (1) 141-6.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19980206
Entered Medline: 19970128

AB . . . prostatic hyperplasia (BPH). An in vitro experiment was carried out using human prostatic carcinoma cell lines (LNCaP, which is androgen **sensitive** and slow growing, and DU145 and PC3, which are androgen insensitive and fast growing), and primary human epithelial and stromal. . . no response to IL-6. The stimulatory effect of CM on prostatic carcinoma cells was significantly reduced by the addition of **anti -IL-6 antibody** to the culture medium. Furthermore, the growth of DU145 and PC3 in serum-free medium was also inhibited by **anti-IL-6 antibody** (P < 0.001). All cell lines tested, except for LNCaP, secreted IL-6 into the culture medium. Results of reverse transcriptase-PCR analysis indicated that IL-6 **receptor** mRNA was present in all carcinoma cell lines but not in epithelial cells or stromal cells derived from BPH. These. . .

L13 ANSWER 4 OF 15 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97129493 MEDLINE
DOCUMENT NUMBER: 97129493 PubMed ID: 8974001
TITLE: The influence of interleukin-6 on the growth of human esophageal cancer cell lines.
AUTHOR: Oka M; Iizuka N; Yamamoto K; Gondo T; Abe T; Hazama S; Akitomi Y; Koishihara Y; Ohsugi Y; Ooba Y; Ishihara T;

Suzuki T
CORPORATE SOURCE: Department of Surgery II, Yamaguchi University School of Medicine, Japan.
SOURCE: JOURNAL OF INTERFERON AND CYTOKINE RESEARCH, (1996 Dec) 16 (12) 1001-6.
Journal code: CD4; 9507088. ISSN: 1079-9907.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970422
Last Updated on STN: 19980206
Entered Medline: 19970410

AB . . . (IL-6). We, therefore, investigated the growth effects ([3H]thymidine uptake assay and direct cell count) of IL-6 on these ECC. IL-6 **receptor** (R) and GP-130 mRNA were detected in all the ECC, using reverse transcriptase-polymerase chain reaction (RT-PCR) assay, and IL-6R was detected in one (YES-3) by immunohistochemical staining. IL-6, **anti-IL-6** monoclonal **antibody** (mAb), or anti-IL-6R mAb caused no reproducible enhancement or suppression of [3H]thymidine uptake by all six ECC. Direct cell count also revealed that the growth enhancement or suppression by IL-6, **anti-IL-6** mAb, or anti-IL-6R mAb was relatively small. Particularly, there was no significant **sensitivity** of YES-3 cells, which definitely produce IL-6 and express IL-6R for IL-6, **anti-IL-6** mAb, or anti-IL6R mAb. These results suggest that some esophageal cancers may produce IL-6 and express IL-6R. However, no major. . .

L13 ANSWER 5 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97093923 MEDLINE
DOCUMENT NUMBER: 97093923 PubMed ID: 8939386
TITLE: Usefulness of flow cytometric detection of cell surface interleukin-6 receptors in human myeloma cell lines.
AUTHOR: Chen Y H; Feng X X; Hagen K
CORPORATE SOURCE: Department of Medicine, College of Medicine, University of Illinois at Chicago, USA.
SOURCE: CLINICAL AND LABORATORY HAEMATOLOGY, (1996 Sep) 18 (3) 161-9.
Journal code: DKF; 7907061. ISSN: 0141-9854.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970313
Last Updated on STN: 19980206
Entered Medline: 19970228

AB A flow cytometric procedure was used to analyse the cell surface interleukin-6 **receptor** (IL-6R) based on the principle of detecting the binding of IL-6 to IL-6R. The bound IL-6 was visualized by reacting with **anti-IL-6 antibody**, a second biotinylated **antibody** to immunoglobulins, fluorescein-conjugated avidin and biotinylated, fluorescein-conjugated bovine serum albumin. Studies with a number of human myeloma cell lines showed. . . as U266-BL cells. The relative IL-6R density of various myeloma cell lines thus determined was found to correlate with their **sensitivity** to the growth inhibitory effect of glucocorticoids in vitro. Quantitatively, calibration of the staining procedure with microbeads that have a defined binding capacity for **anti-IL-6 antibody** allowed calculation of cellular IL-6R density that yielded results close to that reported with conventional radio-ligand binding assay. Similarly, the. . .

L13 ANSWER 6 OF 15 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 95136236 MEDLINE
DOCUMENT NUMBER: 95136236 PubMed ID: 7834629
TITLE: Sensitization of human renal cell carcinoma cells to cis-diamminedichloroplatinum(II) by anti-interleukin 6 monoclonal antibody or anti-interleukin 6 receptor monoclonal antibody.
AUTHOR: Mizutani Y; Bonavida B; Koishihara Y; Akamatsu K; Ohsugi Y;
Yoshida O
CORPORATE SOURCE: Department of Urology, Faculty of Medicine, Kyoto University, Japan.
SOURCE: CANCER RESEARCH, (1995 Feb 1) 55 (3) 590-6.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950314
Last Updated on STN: 19980206
Entered Medline: 19950228

AB . . . activity against renal cell carcinoma (RCC). It has been demonstrated that RCC cells secrete interleukin 6 (IL-6) and express IL-6 **receptors** (IL-6Rs). IL-6 inhibits apoptosis and enhances manganese superoxide dismutase expression. Several anticancer chemotherapeutic agents exert their cytotoxic activity in part. . . to the anticancer agents might correlate with IL-6 expression. The present study tested this hypothesis by examining the effect of **anti-IL-6** mAb and anti-IL-6R mAb on the **sensitivity** of human RCC cells to anticancer chemotherapeutic agents. Treatment of Caki-1 cells with **anti-IL-6** mAb or anti-IL-6R mAb in combination with cis-diamminedichloroplatinum(II) (CDDP) or mitomycin C overcame their resistance to CDDP or mitomycin C. However, treatment of Caki-1 cells with **anti-IL-6** mAb or anti-IL-6R mAb in combination with Adriamycin, vinblastine or 5-fluorouracil did not overcome their resistance to these anticancer agents.. . . (Caki-1/DDP), two other RCC cell lines (ACHN and A704), and three freshly derived RCC cells with CDDP in combination with **anti-IL-6** mAb or anti-IL-6R mAb reversed the resistance to CDDP in all these tumors. We then studied the effectiveness of other platinum derivatives. Treatment of Caki-1 cells with **anti-IL-6** mAb or anti-IL-6R mAb enhanced their **sensitivity** to carboplatin, but not to trans-diamminedichloroplatinum(II). Several experiments investigated the mechanism of the **antibody**-mediated sensitization of RCC cells to CDDP. Incubation of Caki-1 cells with **anti-IL-6** mAb or anti-IL-6R mAb did not change the intracellular accumulation of CDDP. The expressions of the multidrug resistant phenotype (gp170) and c-myc oncogene were not affected by the **antibody**-mediated sensitization. Treatment of Caki-1 cells with the **anti-IL-6** mAb or anti-IL-6R mAb down-regulated the expression of glutathione S-transferase pi mRNA. This study demonstrates that treatment of RCC cells with CDDP in combination with **anti-IL-6** mAb or anti-IL-6R mAb can overcome their CDDP-resistance and that the down-regulation of glutathione S-transferase pi expression by **anti-IL-6** mAb or anti-IL-6R mAb might play a role in the enhanced cytotoxicity obtained.(ABSTRACT TRUNCATED AT 400 WORDS)

L13 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1995:186211 BIOSIS
DOCUMENT NUMBER: PREV199598200511
TITLE: Enhanced **sensitivity** of human renal cell carcinoma cells to cisplatin by **anti-IL**

-6 monoclonal **antibody** or **anti**
-**IL-6 receptor** monoclonal
antibody.
AUTHOR(S): Mizutani, Y. (1); Bonavida, B. (1); Koishihara, Y.; Akamatsu, K.; Ohsugi, Y.; Yoshida, O. (1)
CORPORATE SOURCE: (1) Dep. Urol., Fac. Med., Kyoto Univ., Kyoto 606 Japan
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (1995) Vol. 36, No. 0, pp. 340.
Meeting Info.: Eighty-sixth Annual Meeting of the American Association for Cancer Research Toronto, Ontario, Canada March 18-22, 1995
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English
TI Enhanced **sensitivity** of human renal cell carcinoma cells to cisplatin by **anti-IL-6** monoclonal **antibody** or **anti-IL-6 receptor** monoclonal **antibody**.

L13 ANSWER 8 OF 15 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 94194289 MEDLINE
DOCUMENT NUMBER: 94194289 PubMed ID: 8145045
TITLE: Ciliary neurotropic factor, interleukin 11, leukemia inhibitory factor, and oncostatin M are growth factors for human myeloma cell lines using the interleukin 6 signal transducer gp130.
AUTHOR: Zhang X G; Gu J J; Lu Z Y; Yasukawa K; Yancopoulos G D; Turner K; Shoyab M; Taga T; Kishimoto T; Bataille R; +
CORPORATE SOURCE: Institute for Molecular Genetics, CNRS BP5051, Montepellier, France.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1994 Apr 1) 179 (4) 1337-42.
Journal code: I2V; 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940511
Last Updated on STN: 20000303
Entered Medline: 19940505
AB . . . 1.2 ng/ml for IL-11, LIF, and OM. CNTF worked at high concentrations only (90 ng/ml), but addition of soluble CNTF **receptor** increased **sensitivity** to CNTF 30-fold. The growth-promoting effect of these four cytokines was abrogated by anti-gp130 **antibodies**, contrary to results for **anti-IL-6 receptor** or **anti-IL-6 antibodies**. No detectable changes in the morphology and phenotype were found when myeloma cells were cultured with one of these four. . .

L13 ANSWER 9 OF 15 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 94166108 MEDLINE
DOCUMENT NUMBER: 94166108 PubMed ID: 8120920
TITLE: Effects of combined antigrowth factor receptor treatment on
in vitro growth of multiple myeloma.
AUTHOR: Taetle R; Dos Santos B; Ohsugi Y; Koishihara Y; Yamada Y; Messner H; Dalton W
CORPORATE SOURCE: Arizona Cancer Center, Tucson 85724.
CONTRACT NUMBER: CA37641 (NCI)
CA43043 (NCI)
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1994 Mar 16) 86 (6) 450-5.
Journal code: J9J; 7503089. ISSN: 0027-8874.

.PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940412
Last Updated on STN: 19970203
Entered Medline: 19940407

AB BACKGROUND: Although passive serotherapy for cancer with monoclonal **antibodies** is an attractive concept, it has unfortunately had limited efficacy in clinical trials. An alternative approach to passive serotherapy is targeting cell surface growth factor **receptors** with monoclonal **antibodies**. With some limitations, anti-growth factor **receptor antibodies** can limit cell growth by blocking stimulatory or trophic growth factor **receptors** and by marshaling in vivo antitumor immune responses. PURPOSE: The purpose of our study was to determine the extent to which anti-interleukin-6 (IL-6) and anti-transferrin (Tf) **receptor antibodies**, when used individually or combined, could limit myeloma cell growth. METHODS: The four myeloma cell lines studied varied in IL-6. . . cells and by enzyme-linked immunosorbent assay. For cell growth assays, cell lines were plated with various concentrations of IL-6 and anti-**receptor antibodies** and [3H]thymidine uptake determined after 3 days. Cells were grown in varying concentrations of IgG1 monoclonal anti-Tf **receptor antibodies** E2.3 and A27.15 or **antibodies** PM1, AUK 146-15, AUK 64-7, or AUK 12-20 to the human IL-6 **receptor**-alpha protein. Tf and IL-6 **receptors** were detected by immunofluorescence staining. RESULTS: Using short-term proliferation assays, anti-Tf **receptors** and **anti-IL-6 antibodies** caused dose-dependent growth inhibition of varying degrees, and, in one of three cell lines, a combination of anti-Tf and **anti-IL-6 antibodies** showed supra-additive growth inhibition. IL-6-independent cells were inhibited by anti-Tf **receptor antibodies**, while IL-6-dependent cells were resistant to these **antibodies** but **sensitive** to **anti-IL-6 receptor**. Factor-dependent myeloma cells exposed to either anti-Tf or **anti-IL-6 receptor antibodies** for 48 hours lost colony-forming capability. A combination of anti-Tf and **anti-IL-6 antibodies** increased elimination of colony-forming cells at 24 hours. CONCLUSIONS: Anti-**receptor antibodies** have distinct patterns of myeloma cell growth inhibition and inhibit in vitro growth of factor-dependent myeloma cells. Combinations of anti-growth factor **receptor antibodies** also increase toxicity for IL-6-dependent myeloma colony-forming units.

L13 ANSWER 10 OF 15 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 95035398 MEDLINE
DOCUMENT NUMBER: 95035398 PubMed ID: 7948313
TITLE: Detection of IL-6 and its receptor mRNAs in a megakaryocytic cell line, CMK, by an RT-nested PCR.
AUTHOR: Nakayama K
CORPORATE SOURCE: Department of Laboratory Medicine, Kobe University School of Medicine, Japan.
SOURCE: ANNALS OF HEMATOLOGY, (1994 Nov) 69 (5) 245-7.
Journal code: A2P; 9107334. ISSN: 0939-5555.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19980206

Entered Medline: 19941229

AB . . . CMK, has been proposed by Fuse et al. They detected IL-6 mRNA by means of Northern blotting, but not IL-6 **receptor** (IL-6R) mRNA. I also failed to detect IL-6R mRNA by the same means. In this study I showed the expression of IL-6 and its **receptor** mRNAs by means of reverse transcriptase-nested polymerase chain reaction (RT-nested PCR) with an automated electrophoresis PhastSystem. These two procedures considerably enhanced detection **sensitivity**. The proliferation and the differentiation of CMK cells are augmented by IL-6 and inhibited by **anti-IL-6 antibody**. This study continued the autocrine theory of Fuse et al. in CMK cells. Moreover, this is the first detection of. . .

L13 ANSWER 11 OF 15 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 93373298 MEDLINE
DOCUMENT NUMBER: 93373298 PubMed ID: 8364912
TITLE: Growth inhibition of human lung cancer cell lines by interleukin 6 in vitro: a possible role in tumor growth via an autocrine mechanism.
AUTHOR: Takizawa H; Ohtoshi T; Ohta K; Yamashita N; Hirohata S; Hirai K; Hiramatsu K; Ito K
CORPORATE SOURCE: Department of Medicine and Physical Therapy, Tokyo University School of Medicine, Japan.
SOURCE: CANCER RESEARCH, (1993 Sep 15) 53 (18) 4175-81. Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199310
ENTRY DATE: Entered STN: 19931022
Last Updated on STN: 19980206
Entered Medline: 19931001

AB . . . the proliferation of human non-small cell lung cancer cell lines, as shown by the growth accelerating effect of the specific **anti-IL-6 antibody** as well as the effect of exogenously added IL-6. Moreover, IL-6 can be expressed and released by human lung cancer cells, and these cells had specific IL-6 **receptors** on their cell surfaces, suggesting an autocrine mechanism. The growth-inhibitory effect of IL-6 was additive to that of transforming growth factor beta, and could not be neutralized by the addition of anti-transforming growth factor beta **antibody**. These results suggested that IL-6 may function as another class of autocrine growth-inhibiting factor in the growth regulation of human lung cancer. Relatively lower IL-6 **sensitivity** of these cells than noncarcinogenic human bronchial epithelial cells also suggested that escape from growth regulation by inhibitory factors such. . .

L13 ANSWER 12 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:468356 CAPLUS
DOCUMENT NUMBER: 117:68356
TITLE: Method and kits for determining human interleukin-(IL-6)/receptor by immunoassay
INVENTOR(S): Kishimoto, Chuzo; Honda, Mitsuo; Yasukawa, Kiyoshi; Saito, Takashi
PATENT ASSIGNEE(S): Chugai Seiyaku K. K., Japan; Toso K. K.
SOURCE: Jpn. Kokai Tokkyo Koho, 5 pp. CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 04089568	A2	19920323	JP 1990-204074	19900801
JP 3212597	B2	20010925		

AB The title method involves: reacting test IL-6 **receptor** in a sample with **anti-IL-6 receptor** monoclonal **antibody** to form a complex and quantitating a label bound to the complex. Thus, test IL-6 **receptor** in an IL-6 **receptor** monoclonal **antibody**-sensitized plate was incubated at room temp. for 2-3 h, followed by incubation with biotinylated **anti-IL-6 receptor** polyclonal **antibody**, incubation with streptavidin-.beta.-galactosidase complex, and fluorometric measurement of the bound enzyme activity for the IL-6 **receptor** detn. Kits for the detn. also are claimed. The method is rapid and highly **sensitive** and can be used in the simultaneous detn. of a multiple no. of samples.

L13 ANSWER 13 OF 15 MEDLINE DUPLICATE 10

ACCESSION NUMBER: 93011459 MEDLINE
DOCUMENT NUMBER: 93011459 PubMed ID: 1327800
TITLE: Limited involvement of interleukin-6 in the pathogenesis of lethal septic shock as revealed by the effect of monoclonal antibodies against interleukin-6 or its receptor in various murine models.
AUTHOR: Libert C; Vink A; Coulie P; Brouckaert P; Everaerdt B; Van Snick J; Fiers W
CORPORATE SOURCE: Laboratory of Molecular Biology, University of Ghent, Belgium.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1992 Oct) 22 (10) 2625-30.
Journal code: EN5; 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199211
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19980206
Entered Medline: 19921110

AB . . . interleukin (IL)-6 levels and outcome in clinical sepsis and in related animal models, respectively. In the present study, two monoclonal **antibodies** were used to investigate the contribution of IL-6 in the lethal action of tumor necrosis factor (TNF) and of lipopolysaccharide (LPS) in mice. We studied the potential protective properties of an anti-murine (m) IL-6 **antibody** and of an anti-mIL-6 **receptor antibody**. In controlled experiments, we observed that both monoclonal **antibodies** conferred a dose-dependent protection to a lethal dose of mTNF. Detailed studies with the monoclonal **antibodies** indicate, however, that protection was no longer observed when the mTNF dose was slightly higher than the lethal dose. Likewise, the **anti-IL-6** monoclonal **antibody** protected against injections of LPS at a lethal-dose concentration, but here too failed to protect against higher doses of LPS.

The **anti-IL-6** monoclonal **antibody** was unable to protect against mTNF in mice sensitized by galactosamine, the corticoid **receptor** antagonist RU38486 or human (h) IL-1 beta. Protection did not correlate with the serum concentrations of IL-6. Finally, we demonstrate that hIL-6 injection did not change the **sensitivity** of mice towards mTNF. We conclude that, although IL-6 levels may be of value as a marker for the outcome. . . . but real, contribution of IL-6 in some situations might be due to its ability to

up-regulate the level of TNF **receptors**.

L13 ANSWER 14 OF 15 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 93153383 MEDLINE
DOCUMENT NUMBER: 93153383 PubMed ID: 1493476
TITLE: Constitutive production of the interleukins IL-5 and IL-6 by the lymphoma cell line OCI-Ly 17 derived from a patient with malignant lymphoma and hypereosinophilia.
AUTHOR: Chang H; Jamal N; Wang X H; Minden M D; Messner H A
CORPORATE SOURCE: Ontario Cancer Institute, Department of Bioresearch, University of Toronto, Canada.
SOURCE: LEUKEMIA AND LYMPHOMA, (1992 Sep) 8 (1-2) 97-107.
Journal code: BNQ; 9007422. ISSN: 1042-8194.
PUB. COUNTRY: Switzerland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199303
ENTRY DATE: Entered STN: 19930326
Last Updated on STN: 19970203
Entered Medline: 19930310

AB . . . line termed OCI-Ly17. Cells of the line stained positive for CD2 and CD5 determinants and demonstrated rearrangement of the T-cell **receptor** beta chain. The immunoglobulin heavy chain gene was found to be in germ line configuration. Northern blot studies using probes. . . growth promoting activity in the supernatant was reduced in a dose dependent manner by preincubation with increasing concentrations of anti-IL-5 **antibodies**. The supernatants of the cell line were also tested on the IL-6 **sensitive** human myeloma line OCI-My4 and myeloma colonies grew in response. This stimulatory activity within the supernatant was neutralized by addition of increasing concentrations of **anti-IL-6 antibodies**. Although producing IL-5 and IL-6 constitutively, the lymphoma line did not increase proliferation in response to either interleukin, nor did it show a reduced proliferative rate when **antibodies** to IL-5 or IL-6 were added to the cultures.

L13 ANSWER 15 OF 15 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 90078664 MEDLINE
DOCUMENT NUMBER: 90078664 PubMed ID: 2592570
TITLE: Serum levels of interleukin 6, a potent myeloma cell growth factor, as a reflect of disease severity in plasma cell dyscrasias.
AUTHOR: Bataille R; Jourdan M; Zhang X G; Klein B
CORPORATE SOURCE: Institut National de la Sante et de la Recherche Medicale U291, Montpellier, France.
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (1989 Dec) 84 (6) 2008-11.
Journal code: HS7; 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199001
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19900328
Entered Medline: 19900123

AB Using a specific and very **sensitive** (1 pg = 1 U) bioassay, we investigated the presence of IL-6, a potent myeloma cell growth factor, in the. . . concept. Taken together, this suggests that this cytokine is probably involved in vivo during the progressive phase of MM. Thus, **anti-IL-6** or **anti-IL-**

6 receptor antibodies could be useful as therapeutic agents at this stage of the disease.

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